been described in the literature,\textsuperscript{1,2} this is the first case in which this usually innocuous pleural mass has produced significant morbidity and contributed to a patient's death.

The special features of this case include the demonstration of local extrinsic compression of the airway seen on bronchoscopy, postobstructive pneumonia in the occluded segments, and local pulmonary artery thrombosis in the affected lobe.

Development of obstructive pneumonia in rounded atelectasis is understandable. The mass, composed of intertwined thickened pleura and atelectatic lung, can entrap and extrinsically compress neighboring bronchi. With obstruction comes a breakdown of local antibacterial mechanisms and the development of infection in the airway. In a report by Hillerdal,\textsuperscript{3} two patients with rounded atelectasis died of pneumonia, but the relationship to rounded atelectasis was not mentioned. It is possible that this patient's age and prostatic carcinoma may have increased his susceptibility to infection as well, even though his white blood cell count was normal.

The causes of thrombosis at the site of rounded atelectasis are possibly twofold. First, with kinking of blood vessels secondary to rounded atelectasis, regional blood flow may decrease and the concentration of procoagulants in local vessels may rise high enough to initiate clotting. Second, damage of the blood vessel caused by kinking and distortion may induce the formation of prothrombin activators which initiate the cascade leading to clot formation.\textsuperscript{4} It is possible to see pulmonary vascular thrombosis in association with severe pneumonia. However, the presence of thrombosis only in the atelectatic lobe in this patient makes local alterations related to the pleural mass the more likely cause.

Another interesting facet of this case is the notable absence of any radiographic evidence of small pneumoconiotic opacities, despite the severe parenchymal involvement with asbestosis found at autopsy. The dissociation between the chest radiograph and histology in asbestosis is well described.\textsuperscript{5} Epler et al\textsuperscript{6} have shown that approximately 10 percent of individuals with chronic diffuse infiltrative lung disease have normal chest roentgenograms. That series included six cases of radiographically inapparent asbestosis. Rockoff and Schwartz\textsuperscript{7} have estimated that application of the International Labour Organization classification can result in a 10 to 20 percent probability of a normal radiograph interpretation in cases of histologically significant asbestosis.

Several articles have described the chest radiographic, bronchographic, tomographic and CT appearance of rounded atelectasis.\textsuperscript{8-11} In most cases, the plain film shows a rounded mass within the lower lobe, as was found in this patient. Lateral tomography may show vessels and bronchi near the mass curving toward and converging on the edge of the mass. The CT scan may show a rounded mass, 4 to 7 cm in diameter, most dense at its periphery, which forms an acute angle with the pleura, with pleural scarring thickest adjacent to the mass. Vessels and bronchi may be seen curving toward the mass. Recognition of these features, while not strictly pathognomonic,\textsuperscript{12} can generally forestall invasive testing and surgical intervention.\textsuperscript{13} However, even though rounded atelectasis is generally benign and sometimes associated with spontaneous resolution,\textsuperscript{11,12,14} this case demonstrates how it can secondarily contribute to patient morbidity and mortality.

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Dialysis-induced Respiratory Acidosis*

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The inability to increase alveolar ventilation can lead to CO₂ retention and acute respiratory acidosis in patients with ventilatory limitation. In this case, a young woman receiving maximum ventilatory support was unable to excrete excess CO₂, associated with increasing dianeal concentrations of peritoneal dialysis. Since the patient's lung disease had necessitated a large amount of ventilatory support, the patient was unable to increase $V_{\text{E}}$ appropriately to handle excess CO₂. Peritoneal dialysate was an additional source of carbohydrates. Peritoneal dialysate is an additional carbohydrate source that may result in hypercapnia and respiratory acidosis in patients with respiratory compromise. To our knowledge, this is the first case report in an adult which demonstrates that peritoneal dialysis with high glucose loads produced an acute respiratory acidosis that was reversed by decreasing the glucose concentrations in the dialysate. Excess CO₂ production should be considered with respiratory disorders associated with dialysis. (Chest 1990; 98:1285-88)

**SLE** = systemic lupus erythematosus; **ANA** = antinuclear antibody; **RQ** = respiratory quotient; **TPN** = total parenteral nutrition

The development of acute respiratory acidosis or the inability to wean a patient from mechanical ventilation often results in an assessment of $V_{\text{CO₂}}$. Increased $V_{\text{CO₂}}$ is seen with fever, sepsis, hyperthyroidism, injury, increased muscular activity or excessive use of carbohydrates during enteral or parenteral hyperalimentation.¹ An increase in $V_{\text{CO₂}}$ from excessive carbohydrate metabolism has been associated with CO₂ retention and failure to wean in patients with chronic obstructive lung disease, in those with bilateral empyemas, in those recovering from ARDS, and in patients who have sustained chest trauma.² We present a patient with SLE and ARDS who developed acute respiratory acidosis associated with the use of increased dianeal concentrations in the peritoneal dialysis fluid. The patient was receiving maximum ventilatory support and a decrease in the dianeal concentration resulted in a return to baseline acid-base and ventilatory status.

Peritoneal dialysis solutions can be an additional source of carbohydrate load for patients with limited ventilatory status and can lead to acute respiratory acidosis. The pulmonary complications of peritoneal dialysis include acute pulmonary edema, pleural effusion, basal atelectasis, pneumonia and hydrothorax.³ This case represents yet another complication of this form of therapy in critically ill patients with renal failure.

**Case Report**

A 27-year-old white woman presented with shortness of breath, cough and fever. She had been well until one year prior to admission when she was noted to have hypertension. She subsequently developed symmetrical polyarthralgias in her hands, wrists, knees and ankles. Bilateral lower extremity edema was also noted at that time. She complained of dyspnea on exertion, pleuritic chest pain and a facial skin rash. Evaluation revealed renal insufficiency and hemolytic anemia. A renal biopsy demonstrated necrotizing glomerulonephritis. An ANA titer was 1:640 with a homogeneous pattern, highly suggestive of SLE. Therapy was initiated with prednisone, cyclophosphamide and plasmapheresis. After seven weeks of therapy, she developed increasing shortness of breath, a nonproductive cough and low-grade fever. Bronchoscopy revealed diffuse pulmonary hemorrhage and the silver methenamine stain was positive for *Pneumocystis carinii*. Her oxygenation status deteriorated and she required mechanical ventilatory support. High FIO₂ and PEEP were required to maintain adequate arterial oxygen saturation. Her renal insufficiency subsequently worsened and peritoneal dialysis was begun.

The hospital course was complicated by two episodes of moderate hyperglycemia and acute respiratory acidosis (Table 1). The dianeal concentration in the peritoneal dialysate had increased from 1.5 to 3.0 and 4.25 g percent prior to each of these episodes. The respiratory acidosis resolved each time after the dialysate dianeal concentration was decreased back to 1.5 g percent (Fig 1). The patient was weaned in the volume control mode throughout this time and no significant changes were made in the settings. In the first episode the rate of exchange was 4 L/h. In the second episode the rate of exchange was 3 L/h. As can be appreciated from Table 1, the $V_{\text{E}}$ during this period was approximately 30 L.

Prior efforts to increase the ventilatory support led to the development of "occult PEEP" and resulted in hypotension. She

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**Figure 1.** Respiratory acidosis during periods of increased dialysate dianeal concentration. The arrows show two episodes of respiratory acidosis coincident with the increase of glucose in the dianeal to 3.0 and 4.25 g percent, respectively.
Table 1—Clinical Data for Episodes of Respiratory Acidosis*

<table>
<thead>
<tr>
<th>Day</th>
<th>% Dianeeal in Dialysate</th>
<th>pH</th>
<th>Pco2</th>
<th>Po2</th>
<th>Peak Pressures</th>
<th>Serum Glucose Value</th>
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<td>47.7</td>
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<td>7.29</td>
<td>42</td>
<td>68</td>
<td>80</td>
<td>117</td>
</tr>
</tbody>
</table>

*Mechanical ventilation by Siemens Servo 900C Ventilator (Siemens Elema Corporation, NJ). Ventilator settings throughout were volume control (VC); TV = 750 to 800 ml (set parameters); RR = 34 to 44; PEEP = 5. Range of Fio2, 50 to 90 percent.

also was receiving parenteral nutrition during these episodes, but no change in concentration, rate of infusion, or insulin concentration had been made.

**Discussion**

Peritoneal dialysis has been utilized for the treatment of uremia since the 1920s and is commonly utilized in the care of critically ill patients with renal failure. The metabolic complications of peritoneal dialysis (hyperglycemia, hyperosmolar coma, hypervolemia and hypovolemia) previously have been documented. Pulmonary complications also have been well documented and include acute pulmonary edema, pleural effusions, basal atelectasis and pneumonia. A single case of acute respiratory acidosis was reported by Whang et al., in a seven-month-old boy with chemical burns who developed diffuse pulmonary infiltrates while receiving peritoneal dialysis.

This patient's hypercapnia and acute respiratory acidosis resulting from the increased carbohydrate load associated with peritoneal dialysis. Fluid retention problems necessitated the use of more highly concentrated dialysate exchanges to pull excess fluid out. It has been shown that there is a high correlation between the amount of glucose absorbed during peritoneal dialysis and the concentration of glucose in the dialysate. Others have found that the use of 4.25 g percent glucose concentration resulted in absorption of approximately 25 to 52 g of glucose per liter of dialysate for 3- and 6-h exchanges, respectively. The use of 1.5 g percent resulted in the absorption of 10 and 22 g/L of dialysate for 3- and 6-h exchanges. Glucose absorption is dependent on the volume, dwell time, sclerosis of the peritoneal membrane and presence or absence of acetate in the dialysate.

Studies have shown that patients on peritoneal dialysis using high glucose dehydrating dialysate have increased body fat, accompanied by increases in plasma cholesterol and triglycerides that correlates with the concentration of the glucose in the dialysate fluid. Thus, excess carbohydrate loading in renal failure patients leads to lipogenesis. Conversion of carbohydrate to fat is associated with a RQ of approximately 8.0 reflecting the much greater production of CO2 per oxygen consumed. Elevated CO2 production in normal patients does not result in hypercapnia, since normal patients respond by increasing VE. In this setting of compromised ventilatory status and respiratory muscle function, the patient could not compensate for the increased CO2 load.

There are other possible causes of the patient's respiratory acidosis, including fever secondary to SLE or sepsis. Her fever fluctuated between 36.7° and 38.9° C throughout this two-week period, but did not coincide with the two episodes of respiratory acidosis. An additional factor that contributes to respiratory acidosis is increased muscular activity. Sedation decreased her work of breathing and prevented her from fighting the ventilator. On four of the days evaluated during this period, her respiratory rate did exceed the ventilator rate. This alteration in her alveolar ventilation did not coincide with the hypercapnia.

We believe that excess carbohydrates related to the high glucose concentration in the peritoneal dialysate was responsible for the production of respiratory acidosis in this patient. There have been reports in the literature of patients with COPD or ARDS developing hypercapnia as a result of increased CO2 production from TPN requiring institution of mechanical ventilation or prolongation of weaning. There also have been studies in patients on continuous ambulatory peritoneal dialysis with normal respiratory function who have increased VE that parallels the increase in Vco2 and V02. To our knowledge, this is the first case report in which a patient with preexisting pulmonary impairment could not compensate for the increased metabolic load from peritoneal dialysis. This clinical situation is analogous to the respiratory insufficiency that may result from TPN in patients with respiratory disorders.

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Occult Fatal Pulmonary Embolism with Disseminated Intravascular Coagulation*

An Unusual Case Masquerading As Miliary Tuberculosis

Cheuk-Kai Wong, M.B.; Chu-Pak Lau, M.D.; Chun-Ho Cheng, M.B.; and Wing-Fung Ng, M.B.

We report a fatal case of occult pulmonary embolism complicating bronchogenic carcinoma which presented with rapidly progressive pulmonary miliary shadows and respiratory failure. A clotting profile abnormality compatible with disseminated intravascular coagulation was noted. Postmortem examination showed extensive clots occluding the major pulmonary vessels and areas of pulmonary infarcts. Histologic examination revealed fibrin deposition in the microvasculature compatible with DIC. Cases of pulmonary embolism with DIC have previously been reported, but this is the first case with pathologic confirmation. Thus, unusual presentation with diffuse lung shadow and DIC should not deter the clinician from correct diagnosis so that appropriate treatment can be promptly started. (Chest 1990; 98:1288-90)

DIC = disseminated intravascular coagulation

Although pulmonary embolism is known to be associated with malignancies, the clinical diagnosis of pulmonary embolism is often difficult in a critically ill patient with diffuse lung shadows. However, an early and correct diagnosis is the only way that the prognosis can be improved.

CASE REPORT

A 43-year-old woman was admitted to a chest hospital because of exertional dyspnea and hemoptysis for three weeks. She was a nonsmoker and had no history of tuberculosis. Chest roentgenogram showed bilateral miliary shadows and a right hilar opacity. Sputum smear for acid-fast bacilli was negative. Because of the local prevalence of pulmonary tuberculosis, she was started on antituberculous treatment with isoniazid, streptomycin, and pyrazinamide. Laboratory data were normal except a low platelet count of 59,000/µL. The coagulation profile (prothrombin and partial thromboplastin time) was normal. The sputum cytology study results were negative for malignant cells. She rapidly deteriorated in the next few days with increasing dyspnea, although the hemoptysis had apparently stopped. The platelet count dropped further to 31,000/µL. Five days after admission, she had severe respiratory distress with circulatory embarrassment and was transferred to our intensive care unit. Examination revealed a weak but regular pulse of 150 beats per minute, central cyanosis, respiratory rate of 40 per minute, and a low systolic blood pressure of 85 mm Hg with poor peripheral circulation. The jugular venous pressure was raised to the angle of the jaw. There were petechiae over both legs, and there was no cals swelling or tenderness. Repeated chest roentgenogram showed that the right hilar shadow had enlarged with bilateral diffuse miliary pulmonary infiltrate (Fig 1). An electrocardiogram revealed only a sinus tachycardia of 150 per minute. An echocardiogram excluded any pericardial effusion. In addition, the pulmonary artery and the right heart chambers were enlarged with an impaired right ventricular contraction. The left ventricle was active. A presumptive diagnosis of diffuse pulmonary alveolar pathology with acute cor pulmonale was made. Laboratory result revealed the picture of disseminated intravascular coagulation: platelet count, 8,000/µL; prothrombin time, 46 seconds (control: 12 seconds); partial thromboplastin time of more than 110 seconds (control 28.2 seconds); thrombin time of more than 110 seconds (control 14.3 seconds); fibrinogen, 0.1 g/L (normal 1.40 to 3.38 g/L); and a serum fibrinogen degradation product of more than 200 µg/mL. She was treated with broad spectrum antibiotics, mechanical ventilation for the hypoxic respiratory failure, and inotropic support. However, she rapidly succumbed in the next hour.

Her relatives agreed only to a limited postmortem examination of the lung. The main pulmonary artery was dilated and both the left and right pulmonary arteries were occluded with fresh blood clots which extended to distal vessels. There were patches of wedge-shaped pulmonary infarcts (Fig 2). Both pulmonary parenchyma were filled with miliary tumor nodules, and a tumor mass arising from the anterior segment of the right upper lobe bronchus was found causing distal collapse. Histologic examination revealed

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